Baseline and Modulated Acoustic Startle Responses in Adolescent Girls With Posttraumatic Stress Disorder

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ABSTRACT

Objective: To assess baseline and modulated acoustic startle responses in adolescent girls with posttraumatic stress disorder (PTSD). **Method:** Twenty-eight adolescent girls with PTSD and 23 healthy control girls were recruited for participation in the study. Acoustic stimuli were bursts of white noise of 104 dB presented biaurally through headphones. Baseline startle responses as well as prepulse inhibition, a 1,000-Hz prestimulation tone presented 120 milliseconds before the startle stimulus for 30 milliseconds, and prepulse facilitation, a 1000-Hz prestimulation tone presented continuously for 2, 000 milliseconds before the startle stimulus, were compared in these two groups of girls. **Results:** At baseline and under neutral testing conditions, the magnitude of the startle response (eye blink) did not differ significantly between girls with PTSD and healthy control girls. There were no significant differences in the degree of prepulse inhibition or facilitation between the two groups of girls. **Conclusions:** Unlike combat veterans with PTSD, adolescent girls with PTSD who report exaggerated startle may not have exaggerated baseline acoustic startle responses in the laboratory. Further research should explore whether girls with PTSD demonstrate altered startle responses under stress and/or evidence of other types of psychophysiological abnormalities. *J. Am. Acad. Child Adolesc. Psychiatry*, 2005;44(8):807–814. **Key Words:** posttraumatic stress disorder, acoustic startle, adolescent girls.

The acoustic startle reflex is a widespread, cross-species response to intense and abrupt onset stimuli. In humans, one component of the acoustic startle response is a reflex eye blink that can be elicited with a sudden burst of noise of at least 90 dB. The eye blink component of the acoustic startle response can be easily recorded and hence is one of the most commonly used markers of the startle response.

An exaggerated startle response is a clinical feature often reported by patients with posttraumatic stress disorder (PTSD) and is one of the hyperarousal or Criteria D symptoms of the *DSM-IV* (American Psychiatric Association, 1994) diagnostic criteria for PTSD. However, in the laboratory, studies of baseline acoustic startle responses in subjects with PTSD have been mixed. An exaggerated baseline startle response has been found in Vietnam combat veterans with PTSD (Butler et al., 1990; Orr et al., 1995), in Israeli combat veterans with PTSD (Shalev et al., 1992), and in Gulf War veterans with PTSD (Morgan et al., 1996). Other investigators (Grillon et al., 1996; Orr et al., 1997b) have reported no differences in baseline acoustic startle responses in men with PTSD compared with combat non-PTSD and healthy civilian controls.

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Two studies examined baseline acoustic startle responses in adult women with chronic PTSD. Morgan and colleagues (1997) compared 13 women with histories of PTSD from sexual assault with 16 healthy control women. Women with PTSD had an elevated baseline acoustic startle response. Furthermore, they displayed a greater eye blink response to the startling tones in the left eye compared with the right eye, but this asymmetrical response did not extend to the healthy control women. Metzger and colleagues (1999) studied three groups of women with histories of childhood sexual abuse: women with current PTSD, women with lifetime PTSD, and women with no history of PTSD. At baseline, under neutral testing conditions, there were no significant differences in the magnitude of the eye blink responses between the three groups of women. However, women with PTSD displayed other signs of heightened autonomic responsivity such as greater heart rate responses and slower absolute habituation of skin conductance responses to startling tones compared with the group without PTSD. These findings replicated similar heart rate and skin conductance findings in men with PTSD (Orr et al., 1995, 1997a; Shalev et al., 1992). In summary, a recent review of baseline acoustic startle response changes in subjects with PTSD compared to nontraumatized controls found a medium effect size (Cohen's $d = 0.49$) across eight of 11 studies (Metzger et al., 1999).

Although PTSD in adolescents closely resembles PTSD in adults, relatively little is known about the phenomenology of adolescents' posttraumatic stress symptomatology and even less is known about the pathophysiology of this condition in teens. Adolescent girls are two (Cuffe et al., 1998) to six (Giaconia et al., 1995) times as likely as adolescent boys to develop PTSD after exposure to a traumatic event. We recently surveyed a group of nonpsychiatric-treatment seeking, inner-city adolescent girls attending a medical clinic and found that 14% and 11% of girls met criteria for full and partial PTSD, respectively (Lipschitz et al., 2000). In this sample of traumatized adolescents, the most common symptoms of posttraumatic stress included psychological distress at trauma reminders (75%), active attempts to avoid people or places connected to the trauma (70%), and hypervigilance (63%). Sixty-two percent of this traumatized sample did not endorse an exaggerated startle. Of those girls who met criteria for full/partial PTSD, only 22% endorsed an exaggerated startle. To date, no studies have examined acoustic startle responses in adolescents with PTSD. Moreover, no studies have examined acoustic startle responses in adolescents exposed to multiple forms of family- and community-based violence, common types of trauma for youngsters residing in urban, inner-city environments. Hence, the first objective of this study was to compare acoustic startle responses at baseline in adolescent girls with PTSD with those of healthy control girls. Our participants for this study were girls who resided in inner-city New Haven at the time of the study.

The acoustic startle response also is a sensitive indicator of sensory processing, and several conditions change the amplitude of the startle response. The startle response is systematically modified by brainstem mechanisms when the startle-eliciting stimulus is preceded by another stimulus (the prepulse) presented at an intensity below startle threshold (Hoffman and Ison, 1980). Startle is inhibited by brief (≤20 milliseconds), low-intensity, nonstartling stimuli presented at short intervals (30–240 milliseconds) before the startle stimulus (Harbin and Berg, 1985). This is termed prepulse inhibition (PPI). Conversely, startle is augmented by longer (>1,400 milliseconds) prestimuli that are sustained continuously throughout the prestimulation interval preceding the startle stimulus (Graham et al., 1975). This is termed prepulse facilitation (PPF). In humans, PPI deficits have been associated with perceptual abnormalities, increased anxiety, and difficulty gating intrusive thoughts (Braff and Geyer, 1990). Patients with PTSD report many intrusive and distressing trauma-related thoughts and two previous studies have documented PPI deficits in patients with PTSD. Grillon and colleagues (1996) reported that 21 Vietnam combat veterans with PTSD showed deficits in PPI compared with 10 combat and 17 healthy civilian controls. In the only published study of acoustic startle responses in traumatized children to date, Ornitz and Pynoos (1989) compared the modulation of acoustic startle responses of six healthy children with those of six children, aged 8–13 years, who developed PTSD after exposure to a sniper attack in their school yard. Children with PTSD displayed a reduced level of PPI compared with the control children. In our sample of traumatized, inner-city adolescents girls, 65% reported intrusive thoughts of their trauma, and of those girls with full/partial PTSD, 77% of them reported intrusive thoughts of their trauma in the past month. Thus, the second objective of this study was to compare the effects of

prepulse modulation on the acoustic startle responses of adolescent girls with PTSD with those of healthy, nontraumatized adolescent girls.

METHOD

Participants

Individuals participating in this study consisted of 28 adolescent girls with PTSD and 23 healthy, nontraumatized adolescent girls (healthy controls). Participants ranged in age from 12 to 21 years (mean = 16.5, SD = 2.8) and had a mean of 11 years (SD = 2.3) of education. Forty-nine percent of the girls were African American, 29% were white, 12% were Latino, and 10% were of other or mixed ethnic backgrounds. All participants were recruited from a hospital-based adolescent primary care clinic and via general advertisements posted in Yale–New Haven Hospital. The Yale–New Haven Adolescent Primary Care Clinic provides general medical and reproductive health care to an urban population from inner-city New Haven, CT. Girls in this study were not seeking psychiatric services, and at the time of this study, they were unmedicated and free of illicit substances as determined by history and urine toxicology screens. Girls who participated in this acoustic startle study were part of a larger group of girls (N = 104) from a previous study that examined the clinical and psychosocial correlates of PTSD in urban, inner-city, adolescent girls (see Lipschitz et al., 2000). There were no significant demographic or clinical differences between youngsters who participated in the acoustic startle study compared with those youngsters who did not participate in the study. The research protocol was approved by the Human Investigations Committee of the Yale University School of Medicine. All participants gave written informed consent and were reimbursed financially for their participation.

Procedure

Each participant completed a battery of standardized, self-report questionnaires as part of the screening procedure. These included the Adolescent Dissociation Experience Scale (Armstrong et al., 1997), the Multidimensional Anxiety Scale for Children (March et al., 1997), and the Beck Depression Inventory (Beck and Steer, 1987). A parent completed the Child Behavior Checklist (Achenbach, 1991). Participants were interviewed by one of two master's degree–level research associates for the presence and timing of all *DSM-IV* (American Psychiatric Association, 1994) Criterion A traumas using the PTSD section of the Schedule of Affective Disorders and Schizophrenia for School-Age Children- Present and Lifetime version (K-SADS-PL) (Kaufman et al., 1997a). Current symptoms of PTSD were established with the child and adolescent PTSD checklist (Amaya-Jackson et al., 2000). Next, each adolescent was interviewed with the remainder of the K-SADS-PL to obtain additional comorbid Axis I psychiatric diagnoses. Final psychiatric diagnoses and groupings were determined by a child and adolescent psychiatrist (D.S.L.), an adult psychiatrist (A.M.R.), and a research associate (E.B.) using a best-estimate approach (Leckman et al., 1982), taking into account results of the self-report questionnaires, the K-SADS-PL, and parents' report on the Child Behavior Checklist. The research team was blind to the adolescent's startle data.

Twenty-eight girls were assigned a diagnosis of PTSD. Seventeen girls met criteria for both lifetime and current PTSD, and 11 girls met criteria for lifetime PTSD only. Identifying traumas included sexual abuse/assault (n = 18, 64%), physical abuse/assault (n = 3, 11%), witnessing domestic violence (n = 2, 7%), witnessing community violence (n = 3, 11%), and vicarious trauma (n = 2, 7%). Comorbid current psychiatric diagnoses included major depression (n = 3, 11%), dysthymia (n = 3, 11%), adjustment disorders with depressed mood (n = 2, 7%), other anxiety disorders (such as social anxiety disorder, simple phobia, and separation anxiety disorder, n = 4, 14%), and disruptive behavioral disorders (such as attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder, n = 3, 11%). None of these girls met criteria for a current substance use disorder.

Twenty-three girls without any Criterion A trauma did not meet any criteria for any psychiatric diagnoses and formed the healthy control group.

Psychometrics of Self-Report Measures

The Child and Adolescent Post Traumatic Stress Disorder Checklist (Child PTSD Checklist) (Amaya-Jackson et al., 2000) is a 28-item scale that asks participants to rate the degree to which each of the 17 symptoms of PTSD was present during the past month. This scale is derived from *DSM-IV* (American Psychiatric Association, 1994) criteria and uses a 4-point Likert severity scale from 0 to 3, corresponding to "not at all" to "all the time." This instrument has been tested in various types of adolescent populations and shows adequate sensitivity, specificity, and diagnostic efficiency (Amaya-Jackson et al., 2000).

Depressive symptomatology was assessed with the 21-item Beck Depression Inventory (Beck and Steer, 1987), which measures cognitive, affective, motivational, and somatic symptoms of depression. For adolescent populations, the BDI has an internal consistency of 0.79, a 5-day test-retest reliability of 0.69, and a 0.67 correlation with clinical ratings of depression.

Symptoms of anxiety were assessed using the Multidimensional Anxiety Scale for Children (March et al., 1997), a 39-item, 4-point Likert format screening scale for anxiety in children and adolescents. There are four main factors: physical symptoms, social anxiety, harm avoidance, and separation anxiety. Three-week and 3-month test-retest reliability is excellent and scales have been normed by gender and age.

The Adolescent Dissociative Experience Scale (Armstrong et al., 1997) is a 30-item, 11-point Likert format self-report questionnaire that asks respondents to indicate the frequency with which certain specific dissociative or depersonalization experiences occur.

The instrument has high test-retest reliability, excellent split-half reliability, and good criterion-referenced validity for an adolescent population.

Startle Response

Startle stimuli were 104 dB, zero rise time, 40-millisecond noise bursts presented through stereo circumaural earphones every 18 to 25 seconds. Prestimulation stimuli were a 72-dB, 1,000 Hz, and 100-μs rise and fall time tone. A total of 24 trials total were presented. For these 24 trials, startle stimuli were presented (1) alone, (2) a 1,000 Hz prestimulation tone presented 120 milliseconds before the startle pulse for 30 milliseconds (PPI, 120-millisecond prepulse), and (3) a 2,000-millisecond prestimulation 1,000-Hz tone presented continuously before the startle stimulus

(PPF, 2,000-millisecond prepulse). The experiment started with the delivery of six pulse-alone trials followed by three blocks each containing two pulses alone, two 120-millisecond prepulse trials, and two 2,000-millisecond prepulse trials presented in random order.

The startle response was recorded from the left orbicularis oculi electromyography (EMG) with one electrode placed below the left lower lid margin and a second placed medial to the outer canthus. The raw EMG was AC amplified with filters set at 90 to 1,000 Hz. The raw signal was rectified and smoothed using a Coulbourn contour following integrator with a time constant of 100 μs. The integrator output was digitized to a Pentium desktop computer at a 500-Hz sampling rate. Data were sampled continuously for 200 milliseconds following the startle probe.

Before the procedure, all subjects were familiarized with the laboratory, and electrodes were applied. During the procedure, subjects sat in a quiet, dimly lighted room in front of a computer screen displaying a continuously appearing screen saver depicting star scenes. Subjects were instructed to sit quietly and watch the screen. All subjects completed the experiment.

Data Analysis

Two measurements describe the startle blink response. These are (1) the latency in milliseconds to peak response (defined as the time it takes for the onset of the startle response) and (2) the amplitude of the response in microvolts defined as the peak response minus the average tonic EMG amplitude in microvolts measured 20 milliseconds before the startle pulse. The response to a short or long prepulse was also expressed as the percentage of decrease (PPI [magnitude of unwarned response – magnitude of warned response]/[magnitude of unwarned response]) or the percentage of increase (PPF [magnitude of warned response – magnitude of unwarned response]/[magnitude of unwarned response]). The magnitude of the unwarned response was defined as the mean of the magnitude of the pulse-alone trials in the same block of trials as the prepulse-modulated trial. No decrease with a short prepulse or no increase with a long prepulse was expressed as zero percentage of change.

Individual EMG trials were excluded if the onset of the blink response was earlier than 20 milliseconds. If the peak response was later than 180 milliseconds following the startle pulse, the response to the pulse was set at zero. Similarly, response magnitude was set at zero if the peak magnitude of the blink response was less than the tonic EMG magnitude recorded during the 20 milliseconds before the startle pulse. Of the 1,224 trials for the 51 subjects, 172 (14.1%) trials were excluded because the blink response occurred earlier than 20 milliseconds or set at zero amplitude response because the blink response occurred later than 180 milliseconds. No trial was set at zero amplitude response because the peak amplitude of the blink response was less than the tonic EMG amplitude in the 100 milliseconds before the startle pulse. An average of two to three trials per subject was either excluded or the amplitude of the response set at zero, and there was no difference among the diagnostic groups in the number of trials excluded or set at zero.

Four sets of analyses were conducted using the startle data. The first set of analyses examined the decrease in pulse-alone startle magnitude (baseline startle) across the four blocks, with the diagnostic group as a between-group variable. The second set of analyses examined the latency to response and response magnitude across the pulse alone and 120 milliseconds and 2,000 milliseconds prepulse trials by diagnostic group. The third set of analyses examined the relation between the percentage of inhibition and facilitation of startle response and the diagnostic groups. The fourth set of analyses examined the relation between the magnitude of pulse alone or baseline startle response and subjects' reports of each of the three PTSD symptom clusters (re-experiencing, avoidance, and hyperarousal) as well as their reports of anxiety and depression.

RESULTS

There were no significant differences in ages among the two diagnostic groups (healthy controls = 16.4 years [SD = 3.9]; PTSD = 16.5 years [SD = 1.9] [t = 0.18, $df = 49$, $p = .85$, not significant (NS)]). There were some significant differences in ethnic composition among the two groups of girls ($\chi^2 = 12.65$, $df = 3$, $p = .005$) with a significantly higher percentage of African-American girls in the PTSD group (27%) and a significantly higher percentage of white girls in the healthy control group (52%).

Table 1 shows differences in self-reported psychopathology between the girls with PTSD and the healthy control girls.

Girls with PTSD were significantly more anxious ($t = 3.54$, $df = 48$, $p = .001$), depressed ($t = 5.13$, $df = 49$, $p < .001$), and dissociated ($t = 4.89$, $df = 48$, $p < .001$) than the healthy control girls.

Startle Response

Fifty-seven percent of girls with PTSD reported an exaggerated startle response compared with 4% of the healthy controls ($\chi^2 = 16.2$, $df = 1$, $p < .001$).

Table 2 shows the mean latency and magnitude of startle response by trial type for the two diagnostic groups.

A separate repeated-measures analysis of variance with age as a covariate and diagnostic group as a between group variable was done to examine habituation for pulse alone. There was a significant decrease in the magnitude of response to pulse alone across the experiment ($F_{11,506} = 2.1$, $p = .02$). There were no main effect for diagnostic group on the decrease in the magnitude of response to pulse alone across the trials ($F = 0.34$, $df = 1,49$, p = NS) and no significant effect of age as a covariate.

The difference in latency and magnitude of startle response by trial type and diagnostic group was examined using analysis of variance with age as a covariate, trial type as a within-group variable, and diagnostic group as a between-group variable. There was an overall significant main effect for trial type ($F = 8.2$, $df = 2,96$,

TABLE 1
Self-reported Psychopathology in Girls with PTSD, Trauma Control, and Healthy Control Girls

Measure	Girls With PTSD (n = 28) Mean (SD)	Healthy Controls (n = 23) Mean (SD)	t	p
MASC (anxiety)	46.9 (20.4)	28.8 (13.7)	3.73	.001
BDI (depression)	14.3 (9.2)	3.7 (3.8)	5.51	<.001
ADES (dissociation)	2.4 (1.7)	0.6 (0.6)	5.21	<.001

Note: MASC = Multidimensional Anxiety Scale for Children (raw scores); BDI = Beck Depression Inventory; ADES = Adolescent Dissociation Experience Scale.

p = .001). As expected, mean startle amplitude with a 120-millisecond prepulse was significantly reduced compared with pulse-alone trials, but there was not a significant increase in amplitude with a 2,000-millisecond prepulse (Table 2). There was not a significant main effect for age (F = 0.3, df = 1,49, p = NS) or diagnostic group (F = 0.001, df = 1,49, p = NS) nor was there significant interaction between trial type and diagnostic group for magnitude of response (F = 1.0, df = 2,96, p = NS). In terms of the proportion inhibition or facilitation, on average across the sample, there were a 59% decrease with a 120-millisecond prepulse and a 35% increase with a 2,000-millisecond prepulse. There was no difference in the proportion inhibition or facilitation between the diagnostic groups (Table 2).

The amplitude of the unmodulated baseline startle response did not correlate significantly with re-experiencing symptoms (r = 0.2, df = 47, p = NS), avoidance symptoms (r = –0.03, df = 47, p = NS), or with hyperarousal symptoms (r = –0.15, p = NS). Baseline startle response did not correlate significantly with self-reported startle responses (r = 0.05, df = 48, p = NS). Baseline startle response did not correlate significantly with anxiety (r = 0.01, df = 50, p = NS) or with depression (r = –0.05, df = 51, p = NS). PPI was not significantly correlated with re-experiencing symptoms (r = 0.02, df = 47, p = NS), avoidance symptoms (r = 0.02, df = 47, p = NS), or hyperarousal symptoms (r = –0.15, df = 47, p = NS). PPI also was not significantly correlated with self-reports of anxiety (r = –0.07, df = 50, p = NS) or of depression (r = 0.001, df = 51, p = NS).

DISCUSSION

To the best of our knowledge, this is the first study to examine acoustic startle responses in traumatized adolescent girls. Our findings did not provide evidence of an exaggerated baseline startle response in urban adolescent girls with PTSD. There seemed to be no significant differences in the prepulse modulation of the acoustic startle response between the two groups of girls, and the magnitude of startle responses did not correlate significantly with self-reported hyperarousal symptoms. Our findings are in contrast to some (Butler et al., 1990; Orr et al., 1995; Shalev et al., 1992) but not all (Grillon et al., 1996, Orr et al., 1997b) the studies conducted in male combat veterans with PTSD. They are, however, in keeping with one other published study of acoustic startle response in traumatized women (Metzger et al., 1999). In the only other study of startle responses in a traumatized younger population, Ornitz and Pynoos (1989) reported a reduced startle in a small sample of traumatized school-age children.

There are several possible explanations for our finding of the absence of an exaggerated startle in the present study. There might be age- and developmentally mediated effects of trauma on the startle response that might explain why traumatized children and adolescents with PTSD show no changes in startle compared with healthy control children, whereas traumatized adults with PTSD show exaggerated clinical responses. Recent neurobiological studies of hypothalamic-pituitary-adrenal axis functioning in traumatized children have shown a different range of abnormalities in children compared with studies in traumatized adults. For instance, DeBellis et al. (1999) found elevated levels of urinary free cortisol in traumatized children compared with reduced levels of urinary free cortisol reported in traumatized combat veterans with PTSD (Mason et al., 1986; Yehuda et al., 1991). Kaufman and colleagues (1997b) found an augmented corticotropin response to ovine corticotropin-releasing factor in abused children compared to the blunted corticotropin, response found in combat veterans with PTSD

TABLE 2
Acoustic Startle Response by Diagnostic Group

	Latency to Response (msec)	Response Magnitude (μV)	Proportion of Reduction or Increase With Prepulse
PTSD (n = 28)			
Pulse alone	98.7 (33.0)	12.7 (10.0)	—
120 msec prepulse	88.3 (33.8)	4.9 (5.1)	0.55 (0.3)
2,000 msec prepulse	89.7 (32.4)	12.9 (11.7)	0.46 (1.0)
Healthy controls (n = 23)			
Pulse alone	106.7 (33.3)	14.8 (15.4)	—
120 msec prepulse	86.6 (33.6)	5.1 (9.2)	0.65 (0.3)
2,000 msec prespulse	108.7 (35.1)	11.4 (14.8)	0.21 (0.7)
F statistic (p)			
Age (1,49)	0.81	0.3	
Trial type (2,96)	2.0	8.2**	
Dx group (1,49)	1.2	0.001	1.4[a], 0.9[b]
Trial × Dx group (2,96)	3.9*	1.0	—

Note: Dx = diagnostic.
[a] 120 milliseconds.
[b] 2,000 milliseconds.
* $p \leq .05$; ** $p \leq .01$.

(Yehuda, 1997). Thus, trauma exposure and PTSD development in younger populations may be associated with small startle responses or reduced responsiveness to acoustic startle stimuli, whereas trauma exposure and PTSD development later in life, for example in adults exposed to combat or sexual assault, might be associated with exaggerated startle responses.

An alternative explanation for the absence of an exaggerated startle in our study involves the context of the experimental setting. In humans, the startle reflex is increased by both fear and anxiety (Bradley et al., 1990). Startle is also increased during the anticipation of an aversive event (Grillon et al., 1991) and when elicited in a stressful experimental context (Grillon et al., 1997). Grillon and Morgan (1999) showed that Gulf War veterans with PTSD have heightened startle responses to fear conditioned stimuli (threat of shock) as well as an inability to modulate their startle responses in response to safety cues. They argue that baseline startle was elevated in Vietnam veterans with PTSD in their studies because they were brought into the laboratory (an unfamiliar setting) and told about the threat of shock (an aversive cue) or an injection of yohimbine (an agent that can provoke flashbacks and panic attacks). In our study, we worked hard to make the laboratory a nonthreatening place. The girls were accompanied by a research associate who was well known to them. The laboratory featured a velvet tent with cutouts of stars and a moon. They were told that they would be watching video games. Thus, our baseline startle measurements were conducted in the relative absence of "experimental stress." However, although we tried to decrease the stress of participation in a novel situation, it is unclear whether the internal state of subjects was indeed one of no anxiety. Adrenergic tone might have been increased in our subjects, resulting in all our girls performing poorly and creating a ceiling effect with respect to startle magnitude. Evidence of this possibility is that we could not elicit much PPF in either our healthy or PTSD subjects.

Finally, our findings cannot be explained by the methodological differences in how we elicited acoustic startle responses. We used a 104-dB burst of white noise to elicit our startle response, and a startle paradigm similar to one used by Grillon and colleagues (1997) to test differences in startle responses between children of alcoholics and children of anxious parents. The magnitude of our startle responses of our two control groups also are in keeping with that of several other studies of startle responses in women (Morgan et al., 1997) and in a another group of healthy teens (L. Mayes, personal communication). However, our participants scored very highly on the Multidimensional Anxiety Scale for Children, a self-report measure of trait

anxiety, and this high level of anxiety could have influenced our overall startle results.

Limitations

One limitation of this study is that we did not study a group of girls with PTSD, all of whom had a specific trauma, e.g., sexual abuse or witnessing an episode of community violence. However, the majority of girls with PTSD identified an interpersonal trauma such as sexual assault (64%) or physical assault (11%) as their Criterion A trauma. Eighty-seven percent of these girls had multiple traumas. A second limitation is that we did not measure other psychophysiological indices of arousal states. For example, the study of heart rate and skin conductance responses to startling tones in PTSD might be a more fruitful avenue to explore. Likewise, we may need to test EMG responses to startling tones using a type of fear-conditioning paradigm to detect significant differences in response between girls with PTSD and our comparison subjects. We are currently running a fear-conditioning paradigm that consists of the threat of an air puff to the larynx and hope to test it in a sample of traumatized teens with PTSD.

Finally, it would have been helpful to administer Likert-type scales of state anxiety and other emotions before and immediately after the startle experiment to more accurately determine adolescents' emotional responses to the context of the experiment.

Clinical Implications

The present study is the first to examine acoustic startle responses in traumatized adolescent girls. Unlike some of the previous adult studies in combat veterans with PTSD and in adult women with sexual assault related–PTSD, there does not appear to be an exaggerated startle response in the laboratory in adolescent girls with PTSD. There do not appear to be significant differences in the modulation of acoustic startle responses between girls with PTSD and healthy control girls. The implication of this study is that some of the psychophysiological abnormalities thought to characterize adults with PTSD might not apply to younger populations. Clearly, we need to do more extensive testing of the acoustic startle paradigm in traumatized children and adolescents to investigate this finding further. One future direction might include the use of a fear-potentiated startle paradigm in studies of adolescents with PTSD as well as the study of other psychophysiological indices of arousal such as heart rate and skin conductance responses to startling tones

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